

AskScience Quarterly

October 2014—Issue 1

the brain
chemistry of

Menstruation,

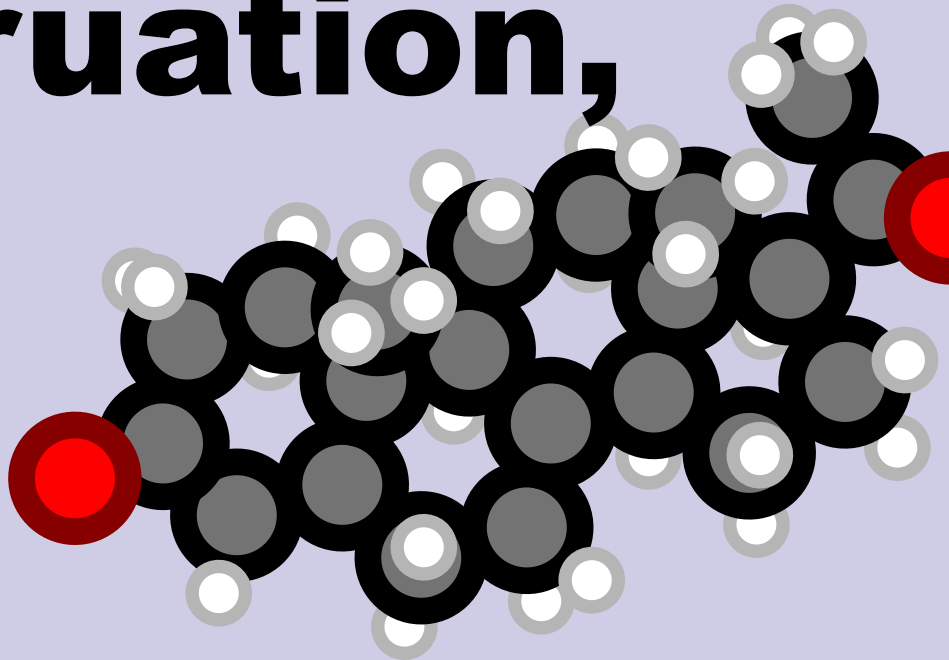
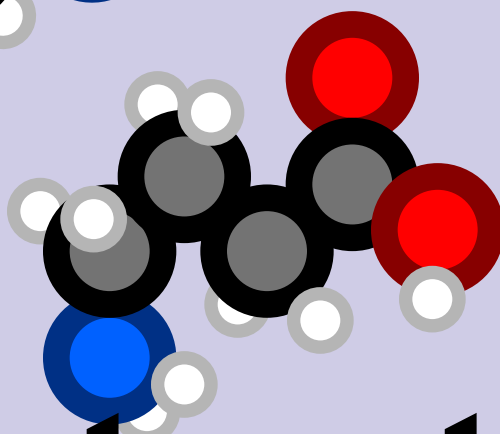
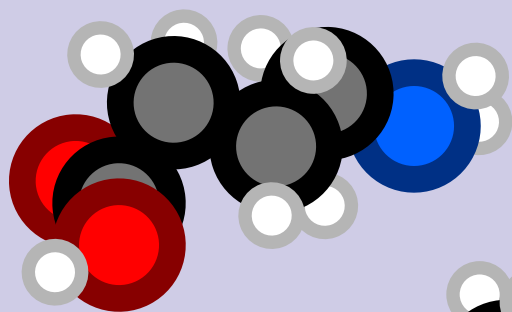
carbon fighting

Algae,

and the human

Eye

in the dark



AskScience Quarterly

Issue 1—Version 34ß

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COVER PICTURE

Neurotransmitter GABA and neurosteroid progesterone molecules outside a phospholipid bilayer.

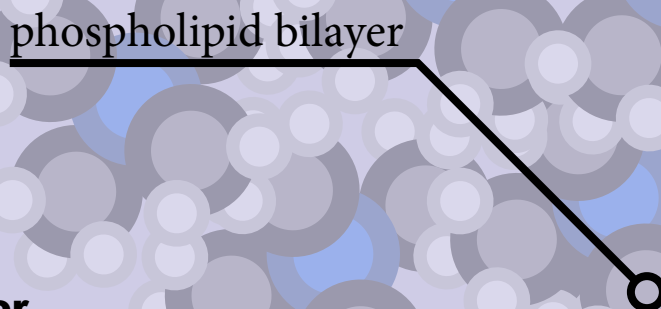
WATERMARK ILLUSTRATIONS

AskScience Group (vectorized by Jesse Griggs)

EDITORS

/u/AsACChemicalEngineer, /u/StringOfLights

phospholipid bilayer



- 3 letter from the editor**
Thoughts from the /r/AskScience Moderation Team
- 4 the last of the dinosaurs**
by /u/StringOfLights
- 5 tiny dinosaurs**
Meet Wallace and Charlie
- 6 what causes the psychological changes seen during pms?**
by Dr. William MK Connelly—Cardiff School of Biosciences, Cardiff University, United Kingdom
- 8 how can algae be used to combat climate change?**
by /u/patchgrabber
- 11 how does the human eye adapt to the dark?**
by Demetri Pananos—Department of Applied Mathematics, Western University
- 13 the fibonacci spiral**
How the Fibonacci sequence and the golden ratio are related
- 14 is mathematics discovered or invented?**
by various authors
- 15 contacts and information**
Want to be an author? Send a letter? Read our copyright license?
- 16 next issue's cover**
Oceans of the Solar System

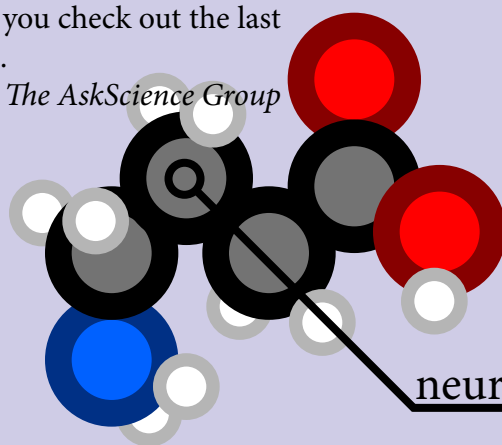
letter from the editor

welcome to the soap-box.

Welcome to the very first issue of the AskScience Quarterly! We are a new popular science magazine created by the scientists of reddit. Specifically, we hail from the subreddit known as AskScience, though some of our writers may come from other communities as well. You can check out our active community by going to www.reddit.com/r/askscience. Our mission is science education and public outreach and while we'll be discussing technical topics, we've always tried our best to keep the language as reader friendly as possible.

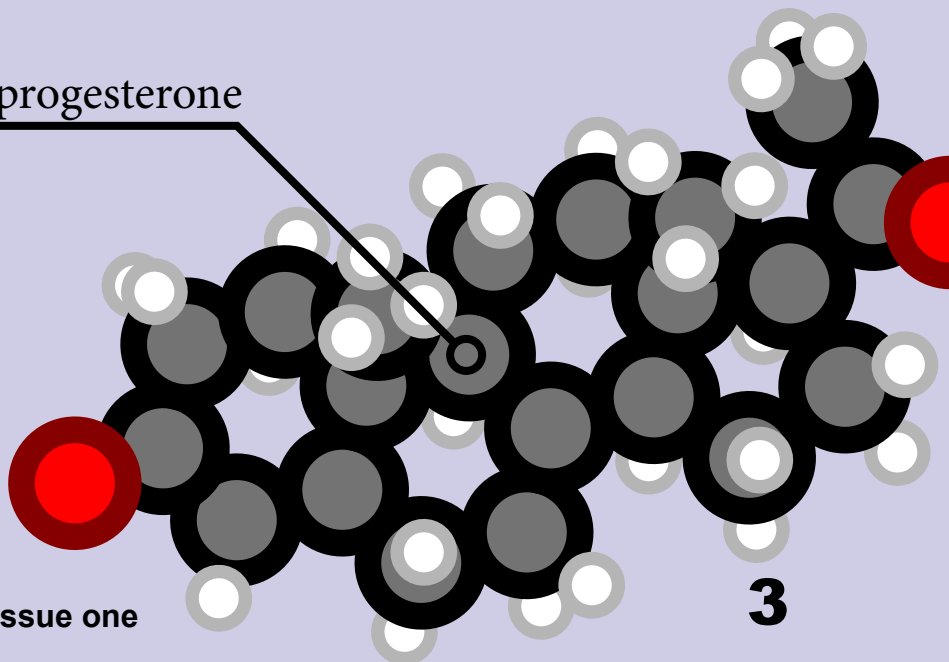
If you've taken the time to read the table of contents, you'll have noticed that we're planning to cover a wide range of topics. We're looking to discuss issues and concepts from all of science. Our articles are written by real scientists who want to explain something *amazing* to everyone, not just other scientists *in the know*. This issue we have dinosaurs, brain chemistry, algae and mathematics, who knows what we'll have next time? Well... If you're interested in a sneak peak, I recommend you check out the last page, but that's just between us.

- The AskScience Group



neurotransmitter GABA

neurosteroid progesterone

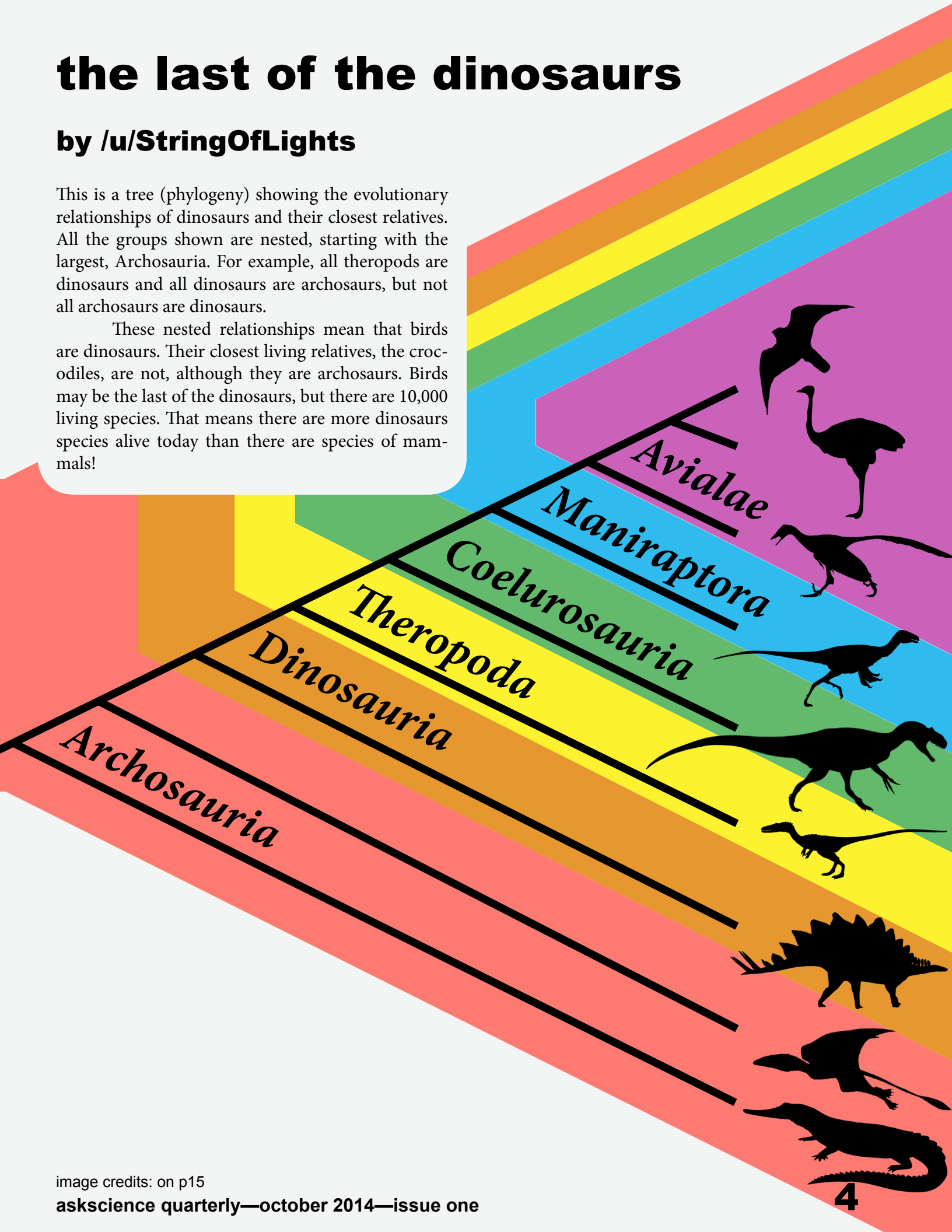


the last of the dinosaurs

by /u/StringOfLights

This is a tree (phylogeny) showing the evolutionary relationships of dinosaurs and their closest relatives. All the groups shown are nested, starting with the largest, Archosauria. For example, all theropods are dinosaurs and all dinosaurs are archosaurs, but not all archosaurs are dinosaurs.

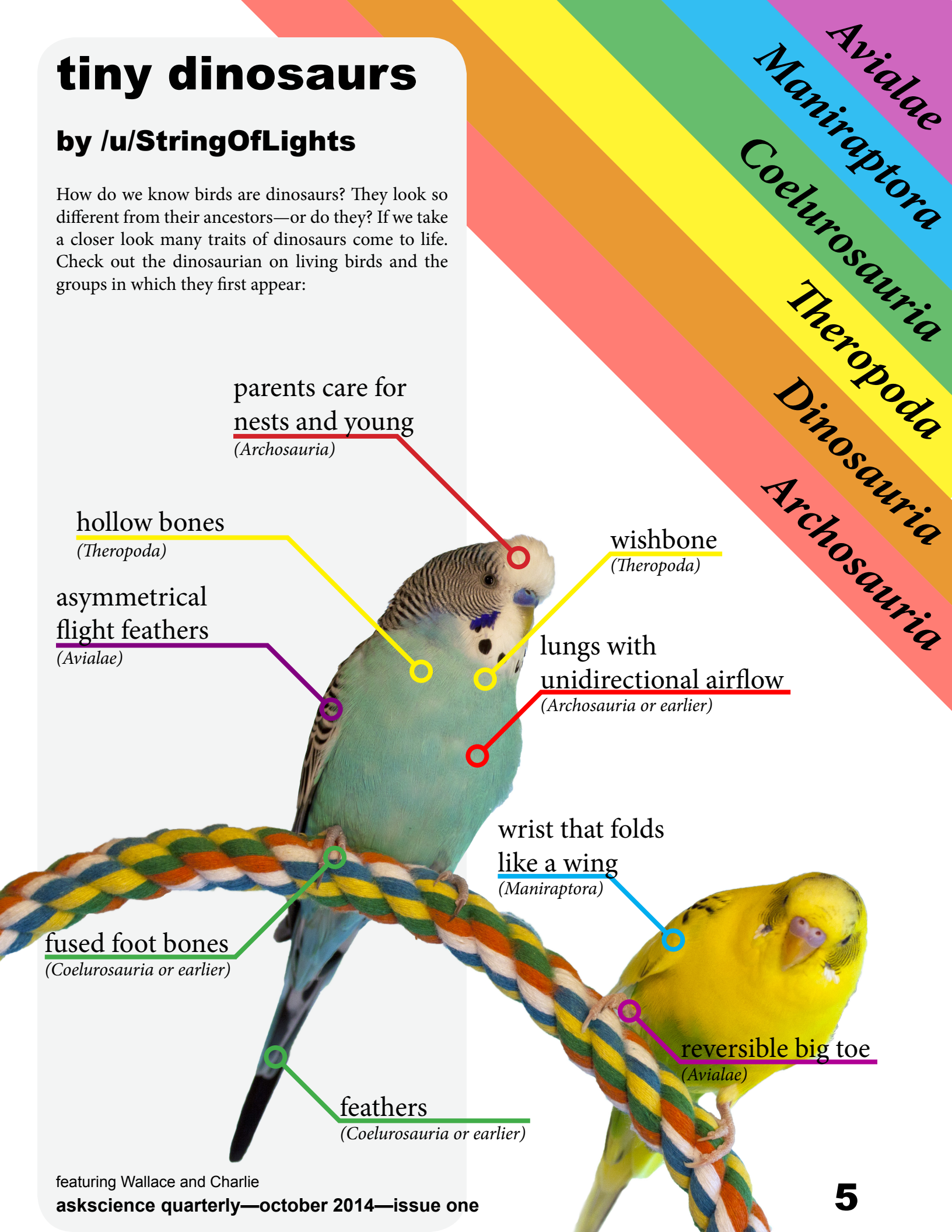
These nested relationships mean that birds are dinosaurs. Their closest living relatives, the crocodiles, are not, although they are archosaurs. Birds may be the last of the dinosaurs, but there are 10,000 living species. That means there are more dinosaurs species alive today than there are species of mammals!



tiny dinosaurs

by /u/StringOfLights

How do we know birds are dinosaurs? They look so different from their ancestors—or do they? If we take a closer look many traits of dinosaurs come to life. Check out the dinosaurian on living birds and the groups in which they first appear:



parents care for
nests and young
(Archosauria)

hollow bones
(Theropoda)

asymmetrical
flight feathers
(Avialae)

wishbone
(Theropoda)

lungs with
unidirectional airflow
(Archosauria or earlier)

wrist that folds
like a wing
(Maniraptora)

fused foot bones
(Coelurosauria or earlier)

feathers
(Coelurosauria or earlier)

reversible big toe
(Avialae)

what causes the psychological changes seen during pms?

by Dr. William MK Connelly

In order to understand what causes PMS, first we need to define what we're actually talking about. PMS, or *premenstrual syndrome* is a collection of largely psychological symptoms that occur in a cyclical fashion, and are seen during the "luteal" phase, that is, the time approximately two weeks before menstruation begins. PMS comes in a more severe form, which I'm going to focus on, called *premenstrual dysphoric disorder* (PMDD) which may affect between 2 and 18% of women and features anger, depression, anxiety and panic attacks.

so what causes pmdd?

It is hard to rule out the idea that some of the psychological changes seen as a result of PMDD are in response to the physical effects that are coincident with the premenstrual phase, as for instance, women with painful periods are more likely to report premenstrual depression [1]. However, it seems almost certain that there is something fundamental going on in the brain during the menstrual cycle, and some of these seem like prime candidates for the causes of PMDD.

Let me point out something interesting: About 40% of women with epilepsy have something called catamenial epilepsy, that is, the frequency of their seizures follows their menstrual cycle, with the highest rate of seizures seen during menstruation [2].

why point out epilepsy?

First, it seems hard to believe that epilepsy could be the result of the physical symptoms seen during the premenstrual phase, this shows that something very significant is changing in the brain. Second, it allows me to tell you another important fact: the hormone progesterone (the blood concentration of which increases over 10 fold during the luteal phase) is a potent anaesthetic when injected into the blood stream, and is able to reduce the occurrence of catamenial epilepsy in about 75% of sufferers [3]. So we can paint a simple picture (i.e. it's not the whole story, but a good start) that catamenial epilepsy is largely caused by the reduced progesterone levels seen when the luteal phase ends, and the menstrual phase begins.

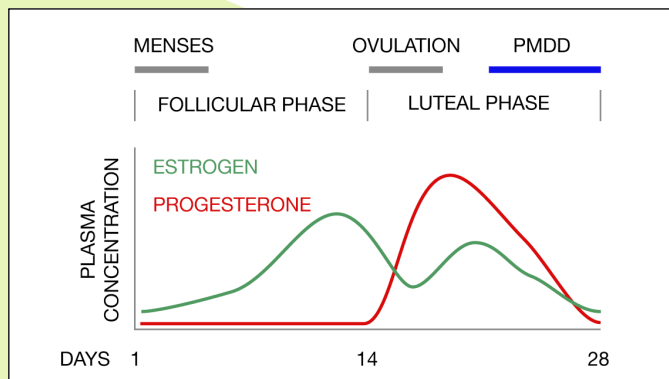


Figure 1. Hormone changes during a menstrual cycle. PMDD is most likely to occur during the late part of the luteal phase, when progesterone levels fall.

why is progesterone important in preventing epilepsy?

Well I need to tell you a few more things. First, I sort of lied to you before when I said progesterone was anti-epileptic and an anaesthetic, it's actually largely inactive, but is converted into something called *allopregnanolone* (ALLO) which really does the work. Though to be fair, ALLO might further be converted into something called 3 α ,5 α -Tetrahydrodeoxycorticosterone.

The conversion of progesterone isn't the important part, what is important is that the compound is metabolised at all. This shows that the brain innately is able to deal with these type of compounds and the reason for this is because the brain is already involved in their production and use, neuroscientists call such compounds "neurosteroids". We don't know how the release of these neurosteroids is regulated, but we know they do *something*, as when you prevent the production of neurosteroids (but leave steroid function in the rest of the body alone) animals get more anxious, showing us neurosteroids are constantly working to keep us calm [4].

Thankfully, we know how these neurosteroids work: They increase the action of proteins called GABA_A receptors. GABA is the primary inhibitory neurotransmitter in the brain. Basically every neuron in the brain expresses GABA_A receptors, allowing GABA to regulate those neuron's activity. GABA_A receptors are targeted by drugs like alcohol and valium, both drugs

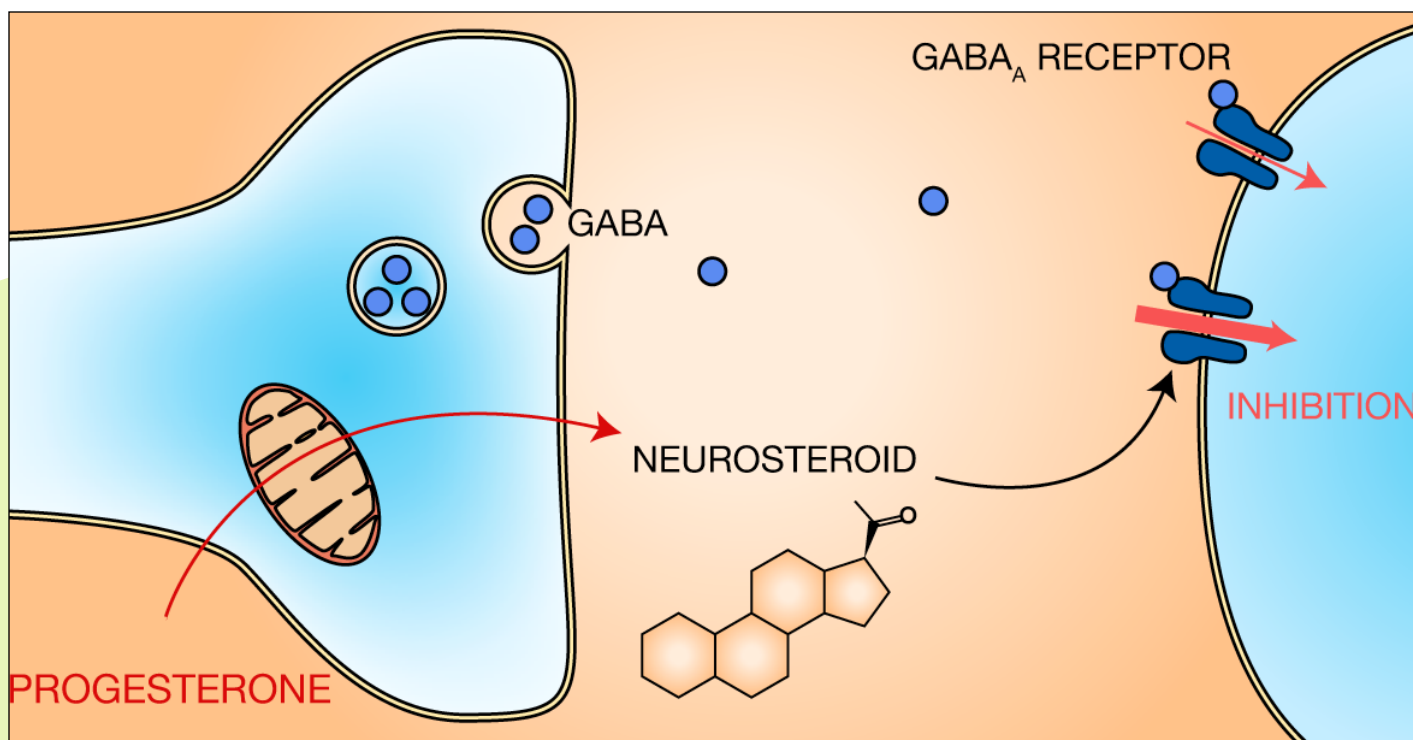


Figure 2. Neurosteroid action at the synapse. GABA is released and acts at GABA_A receptors. Neurosteroids, which are produced from progesterone, act at GABA_A receptors to enhance the inhibitory action of GABA_A receptors. While represented in this fashion for simplicity, neurosteroids may or may not be released by the same neurons which release GABA.

which enhance the action of GABA at this receptor, an effect shared by neurosteroids. This makes it even more clear why catamenial epilepsy occurs; progesterone (through one of its metabolites, like ALLO) enhances the action of GABA, increasing neuronal inhibition and preventing epilepsy. However, at the end of the luteal phase, when progesterone levels fall, GABA no longer produces strong enough inhibition, and seizures can happen.

how does gaba relate to pmdd?

Well now we can put it all together. We know that PMDD happens during the luteal phase, and is associated with anger, anxiety and depression. We know that during the luteal phase, progesterone levels increase and we know that progesterone has anti-anxiety, and anaesthetic actions thanks to the fact that it enhances the action of GABA at GABA_A receptors.

So you're smart reader, can you guess what is happening? I bet you can, and so did a lot of scientists. They guessed that women with PMDD somehow had low progesterone during the luteal phase. However, this does not appear to be the case as progesterone therapy has almost no effect on PMDD and importantly, there appears to be no correlation between plasma progesterone concentrations and PMDD [5, 6].

So I propose an alternative conclusion: If the problem isn't with the progesterone, then what if the

problem is with the GABA_A receptors that would sense the progesterone? Indeed, evidence is mounting that the anxiety that is often seen after pregnancy and associated with puberty may be due to alterations in GABA_A receptors, specifically, that they stop being sensitive to neurosteroids [7, 8]. And it appears that there is a clear regulation of the GABA_A receptors during the menstrual cycle as well [9].

Thus, it appears that many of the symptoms seen during PMDD may be due to a change in inhibitory GABA_A receptors, such that they are no longer sensitive to the neurosteroids that enhance their action. We haven't proved this yet, as it is hard to measure what kind of GABA_A receptors are being expressed in a person's head, nor do we have good animal models of PMDD. However, the clear link between neurosteroids, behaviour, and the menstrual cycle make this one of the leading theories [10].

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how can algae be used to combat climate change?

by /u/patchgrabber

Algae are some of the most efficient organisms on the planet, and they are responsible for 20-30% of the world's primary productivity [1]. Their natural affinity for CO₂ makes them ideal for absorbing the waste gas from industry, and turning it into the oxygen we breath. The resulting biomass isn't wasted either and can undergo further processing to be sold as biodiesel, nutraceuticals, or fertilizer. Additionally algae are not a food crop, and do not compete for arable land that could otherwise be used for food production. All of this makes algae ideally suited for the task of helping to mitigate anthropogenic CO₂.

how does algal co₂ sequestration work?

The first step involves the flue gas itself, which contains the carbon dioxide produced from industry. Here we have a few options for when to capture the CO₂, such as post-combustion, pre-combustion, and oxyfuel combustion. Algal sequestration would be best suited to take place at locations such as power plants, cement plants, or other places of industry that have large outputs of flue gas. These locations typically use post-combustion, which involves capturing the CO₂ after the burning of the fossil fuels. The capture of the gas must be done before the gas can expand to atmospheric pressure as the CO₂ concentration drops rapidly as it travels further from the point source. Flue gas typically has CO₂ concentrations of around 15% [2] and a temperature of 40°C, which is higher

than most algae like, so finding strains tolerant to the high CO₂ and temperatures is a must.

The gas can then be directed to the algal system for sequestration. Two options are favoured for algal culturing, open pond systems and photobioreactor systems. Open pond systems are cheap, but suffer from contamination problems and excessive space requirements. Photobioreactors such as the biofence (pictured on the next page) allow for more control and sterility, but are energy intensive and thus more costly. At pH ≥ 7 the CO₂ typically exists in water as bicarbonate. This is perfect for algae who also like this pH, because they can actively transport the bicarbonate into their cells [3] which the chloroplasts then convert back into CO₂ for the Calvin Cycle.

are there components other than co₂ in flue gas?

Yes. Flue gas is not only composed of CO₂, some other chemical species of importance are NO_x and SO_x gases. While these gases are dangerous to algae in high concentrations, they also contain the nitrogen and sulfur that algae need to grow, and this is the focus of my current research. While even relatively small concentrations of NO_x can inhibit algal growth [4], the combined effect with CO₂ and O₂ can offset the inhibition and promote growth [5]. Sulfur oxides, specifically SO₂, can be problematic if their concentrations are too high, so depending on the algal species the gas may need some pretreatment, although this increases costs.

why isn't the technology more developed?

There was a huge push in the late 1970s by the Department of Energy in the US to find strains of algae suitable for biofuel production. While this isn't directly related to CO₂ sequestration it underscores the problems facing algal energy research, namely, that research funding is related to the price of oil, gasoline and energy.

Back in the gasoline shortage of the '70s, there was a lot of thought as to alternatives, and algae looked promising. As the price of gasoline dropped, so did the funding. Recent initiatives to protect the environment such as the Kyoto Protocol also increased interest in algae research, so it was a mix of cheaper fossil fuels and a lack of desire or planning to reduce pollution that has set back research.



Figure 1. Two rows of biofence housing algae. The biofence is being illuminated with artificial lighting.

why not just store industrially produced CO₂ underground?

Sustainability for one. There are also a variety of potential social problems with sequestering CO₂ underground, and the issue of safety is a big concern for geological sequestration; algae has no real issue with safety. There is also no need to transport the CO₂ to a storage site as all of the algal sequestration is done at the source. The additional economic bonuses brought in by downstream processing also have an advantage as there is no monetary benefit to storing CO₂ underground.

what challenges do algae face?

There are several. Finding the appropriate algal species is a challenge, none of them really have all the characteristics we're looking for. We need high growth rates, tolerances to CO₂, high temperatures and other gases like NO_x as well as the high production of oil in the form of triacylglycerols, which is necessary for biofuels.

We've kept looking though, and several labs have libraries of hundreds to thousands of strains. Genetic engineering of algae is slow, but there are promising genes in yeast, for example, that increase oil production but each one of those yeast genes share ancestry with multiple genes in algae so it is much more complicated to control.

The toxicity of flue gas is another issue that can be handled in several ways such as pretreating the gas, but getting the algae to utilize as much of the NO_x and SO_x as possible is a challenge because they don't grow on it directly and need to be supplied an additional nitrogen source; this can be handled by using municipal wastewater as the growth medium

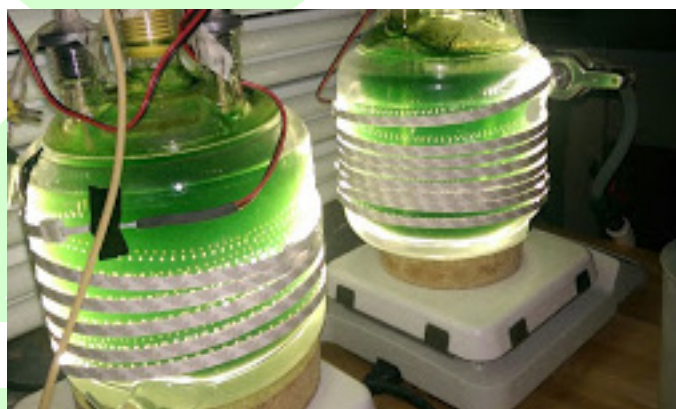


Figure 2. Chemostats are constant volume bioreactors that allow precise control over the growing conditions while continuously adding and removing growth medium. Here we are trying cheaper light sources such as LEDs of varying colors to investigate the effect on growth.

which is high in nitrates, phosphates, all the things algae needs.

Lastly, there is cost. Photobioreactors, flue gas pretreatment or scrubbing, and the cost of downstream processes like converting algae into biodiesel all factor in comparison to current practices. There is also a cost associated with the requirement of inorganic nutrients to feed the algae and the intensive energy needed to cultivate, harvest and dry the biomass produced [6].

what does the future of algal CO₂ sequestration look like?

The future is very promising! Many countries are working on projects using algae as a mitigator of CO₂, and plenty of labs are doing research into the algae themselves. We've found several species of algae that have high extracellular carboanhydrase activity, which is essential for converting carbonate to free CO₂ so the cell can use it. We're also using chemostats to investigate other possible sources of lighting to finely control growing conditions to optimize biomass production.

As we refine our methods for producing biodiesel from algae and find strains with the fatty acid profiles best suited for fuel, we can help alleviate some of the costs associated with using algae systems by allowing industry to profit from selling a cleaner fuel that is made with organisms they already have and care for. Current operations sequestering CO₂ with algae can capture 80-90 tonnes of CO₂ per acre per year, and as the technology develops those numbers will increase.

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how does the human eye adapt to the dark?

by Demetri Pananos

It has happened to all of us before; you are walking home on a bright winter day with the sun reflecting off the fresh snow. You open the door to your house and upon entering: You've lost your sight! Some time later, your eyes adjust to the darker environment and your sight is as it was before. The sudden loss, and gradual restoration, of visual sensitivity following intense illumination is known as dark adaptation. To understand how and why our visual sensitivity is restored, we have to understand the,

1 Rhodopsin lives in the outer segments of the eye's photoreceptors. Under heavy illumination, the photons break up Rhodopsin into its two constituent molecules: Opsin and 11-Cis-Retinal.

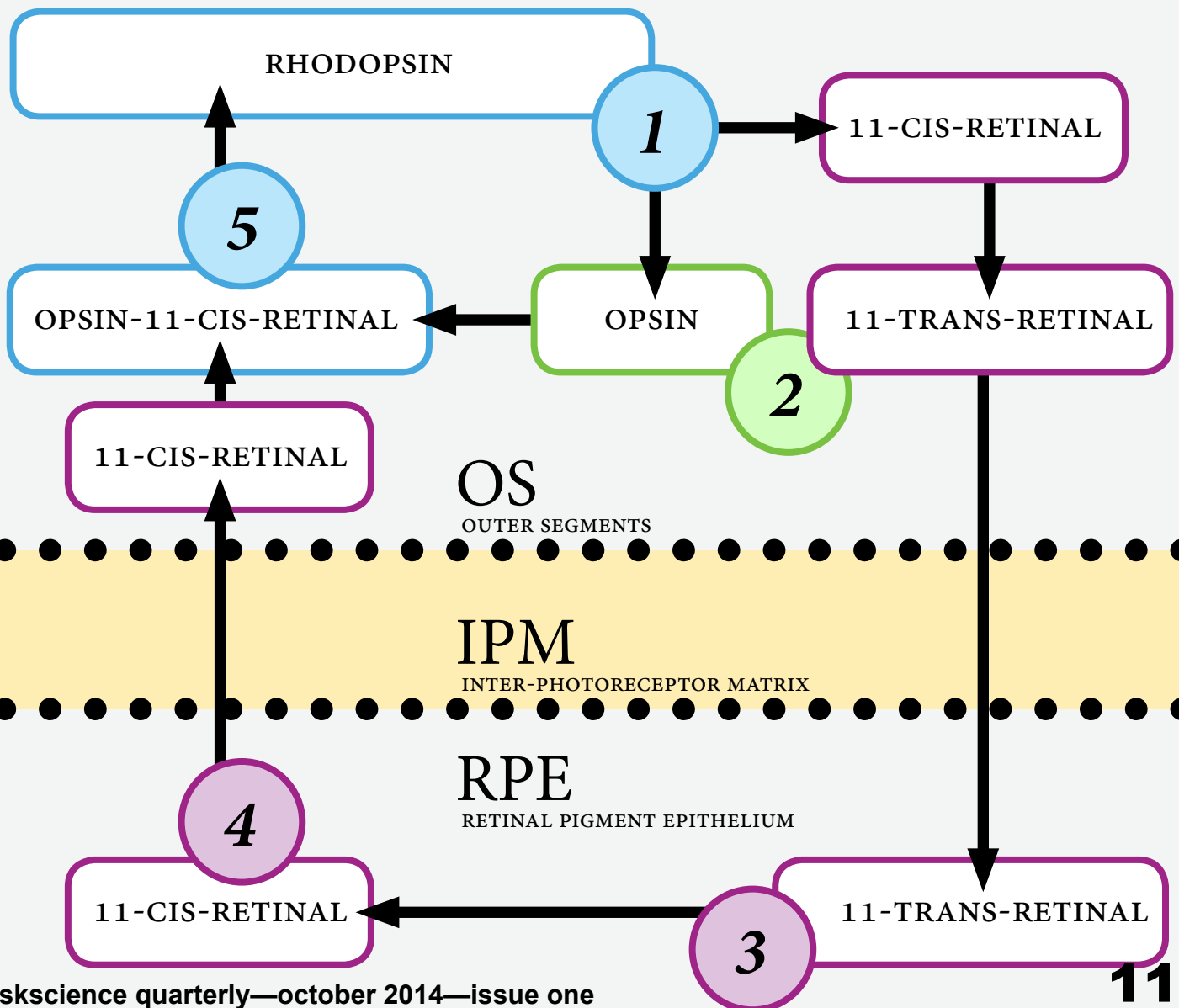
2 A chemical reaction causes 11-Cis-Retinal to change its structure, straightening the molecule out into 11-Trans-Retinal. The Opsin stays in the outer segments of the photoreceptor while the 11-Trans-Retinal is carried across the Inter-Photoreceptor Matrix (IPM) into the Retinal Pigment Epithelium (RPE).

3 To create more Rhodopsin, we need 11-Cis-Retinal. In the RPE, 11-Trans-Retinal is re-bent into 11-Cis-Retinal in preparation to be sent back across the IPM to combine with Opsin.

4 When the illumination ends (e.g. when you walk inside the building) the new 11-Cis-Retinal is pumped back across the IPM where it can bind with free Opsin to create an Opsin-11-Cis-Retinal compound.

5 This Opsin-11-Cis-Retinal compound is then turned into Rhodopsin, and the process starts again once we walk outside.

opsin cycle of vision



That last two parts (4, 5) are where the adaptation happens. As the Opsin combines with the 11-Cis-Retinal, Rhodopsin is created and you gradually regain your visual sensitivity.

how long does it take to get my vision back?

To answer that, we have to use something few biologists use; we have to use mathematics! We want an equation that can tell us how much Rhodopsin is present when I put in a particular time. The easiest way to do that is with a differential equation. Differential equations, or DEs for short, allow us to relate how fast something changes to how much of it is present. So by knowing how quickly the Rhodopsin is being created, we can know how much Rhodopsin is present in the outer segments of the photoreceptors at any time. As scientists do, let's make a few small assumptions:

First, let's assume that as soon as the 11-Cis-Retinal enters the outer segments, it is immediately picked up by a molecule of Opsin. This is called *The Quasi Steady State Assumption*, and is used by Biochemists in modelling enzyme-substrate interactions [1]. Second, let's assume that the rate at which 11-Cis-Retinal is being pumped across the IPM is proportional to the concentration of 11-Cis-Retinal in the RPE. More 11-Cis-Retinal means a faster rate of delivery, while less 11-Cis-Retinal means a slower rate of delivery.

With a few other assumptions regarding the concentration of Opsin in the outer segments, as well as some assumptions on how fast the Opsin-11-Cis-Retinal compound is turned into Rhodopsin, we can create our differential equation, solve it, and have our equation. The details of the mathematics can be found in *Dark Adaptation & The Retinoid Cycle of Vision* [2]. Here it is the final solved equation we'll use:

$$Rh(t) = 1 - K_m W\left(\frac{B}{K_m} \exp\left(\frac{B}{K_m}\right) \exp\left(\frac{-t}{\tau}\right)\right)$$

If you have ever taken an introduction to calculus, or have seen differential equations before, you'd know enough to get this far by yourself! The K_m , B and τ are all parameters, and may differ from person to person. The W is *The Lambert W Function* and is a very special function, much like the logarithm. With the parameters set as $K_m = 0.05$, $B = 1$ and $\tau = 0.5$ we get the curve shown in *Figure 1*. After 10 minutes of being in the dark, Rhodopsin concentration reaches

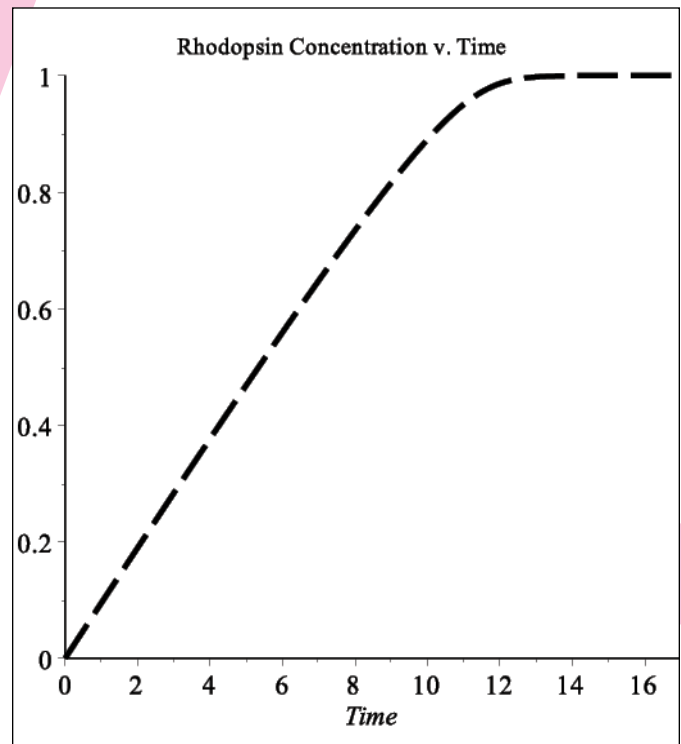


Figure 1. A plot of our equation. On the x-axis is the time after the illumination has ended (in minutes), and on the y-axis is the concentration of rhodopsin (as a fraction).

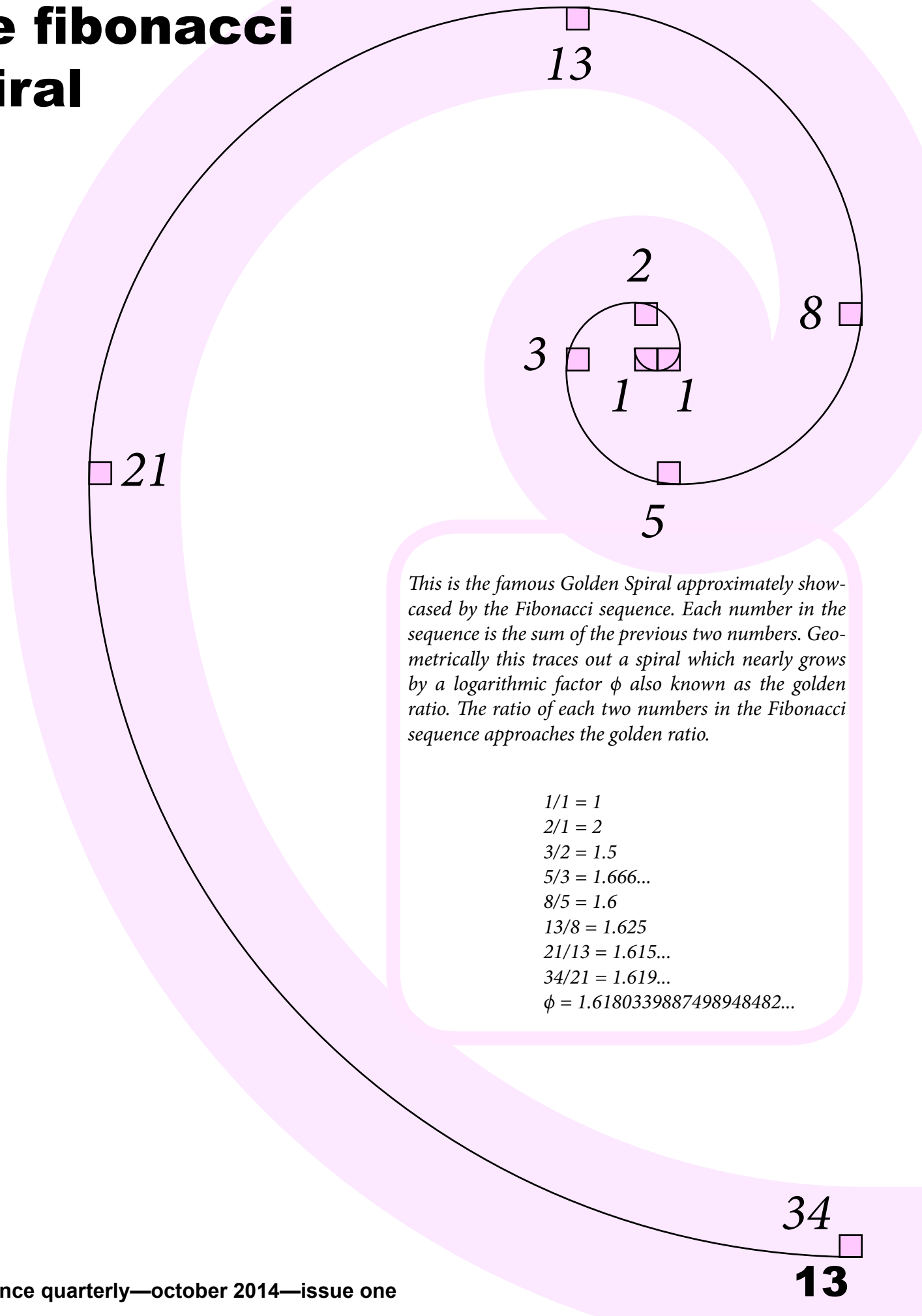
88%, and just after 12 minutes, Rhodopsin concentrations reach 99%. This replenishment of Rhodopsin is what ultimately brings back your vision.

With just a little bit of math, we can quantify how quickly we can adapt in the dark. Furthermore, if we can choose parameters from experiment such that the model matches recovery times for patients with diseases such as Oguchi disease, the mathematics could help pinpoint which step in the Opsin cycle is causing the abnormally slow dark adaptation, furthering our understanding of the disease.

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the fibonacci spiral



This is the famous Golden Spiral approximately shown by the Fibonacci sequence. Each number in the sequence is the sum of the previous two numbers. Geometrically this traces out a spiral which nearly grows by a logarithmic factor ϕ also known as the golden ratio. The ratio of each two numbers in the Fibonacci sequence approaches the golden ratio.

$$1/1 = 1$$

$$2/1 = 2$$

$$3/2 = 1.5$$

$$5/3 = 1.666...$$

$$8/5 = 1.6$$

$$13/8 = 1.625$$

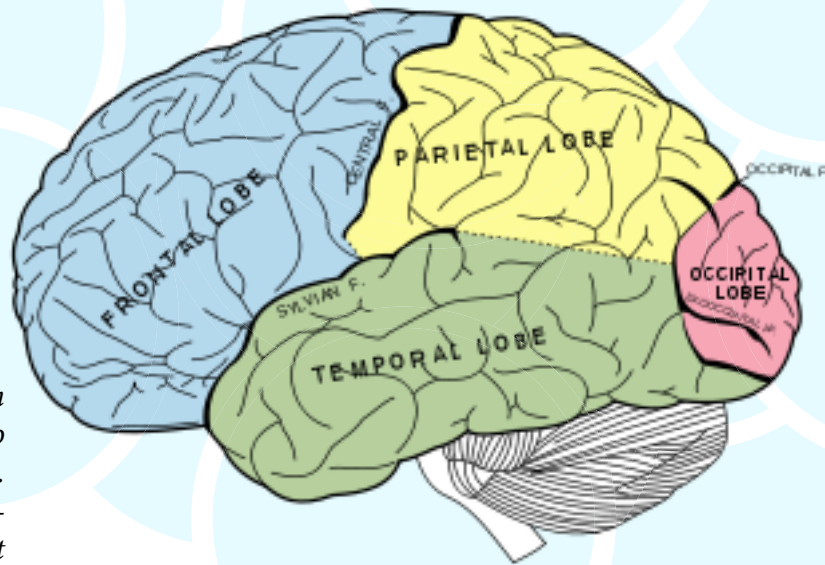
$$21/13 = 1.615...$$

$$34/21 = 1.619...$$

$$\phi = 1.6180339887498948482...$$

is mathematics discovered or invented?

Carl Friedrich Gauss called mathematics the “Queen of the Sciences”. Was he right? You may be surprised to learn that there is no single consensus among scientists. This month’s discussion focuses on the philosophical debate on whether or not mathematics is an intrinsic part of our universe or a useful fiction...



I am normally inclined to say that mathematics is discovered. Axioms are laid down, and we discover their implications. But the nature of things like the fourier transform, which is largely for applications (though not entirely so), seems to make me think that it is somehow invented.

Demetri Pananos

Mathematics is generally considered to be deductive. By analogy then, in the syllogism:

Socrates is a man

Man is mortal

Socrates is mortal

What would we term the conclusion? Certainly more of a discovery than an invention. [...] But then of course, a lot of *applied* maths isn't really like anyway. Instead it might, for example, be more about *inventing* better algorithms or techniques. I'd say that a Fourier Transform fits more into this camp.

/u/petejonze

I am personally far into the Invented camp [...] To me, math is an art above all else. It is an art where you are able to freely explore abstraction and create new ideas. [...] Your work is, of course, criticized by your peers but this criticism is closer to what happens in the artistic community where it's value is based on whether or not it is relevant and interesting.

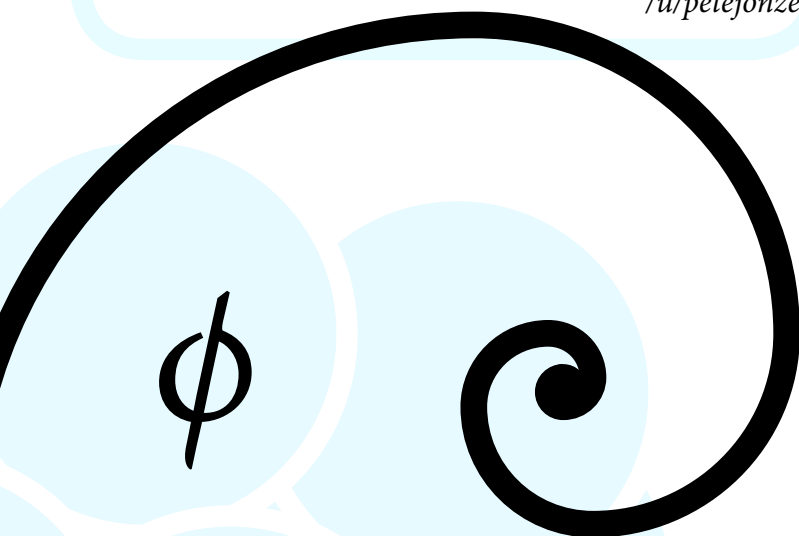
/u/functor7

It's perfectly reasonable to say that Napier and Bürgi independently invented the logarithm operator [...] Mathematicians like Euler then discovered things about that operator that revealed it to have profound implications, allowing the remarkable and unexpected union of arithmetic and geometry. [...] You're free to invent any mathematical toys and tools you like, but you aren't free to assert what is and is not true about those inventions.

Greg Jensen

Evidently (and my experience backs this up), there is not a consensus, but neither is there a right answer to achieve a consensus on; it depends on how you view the nature of math. Personally I fall in the “discover” camp.

/u/diazona



contacts and information

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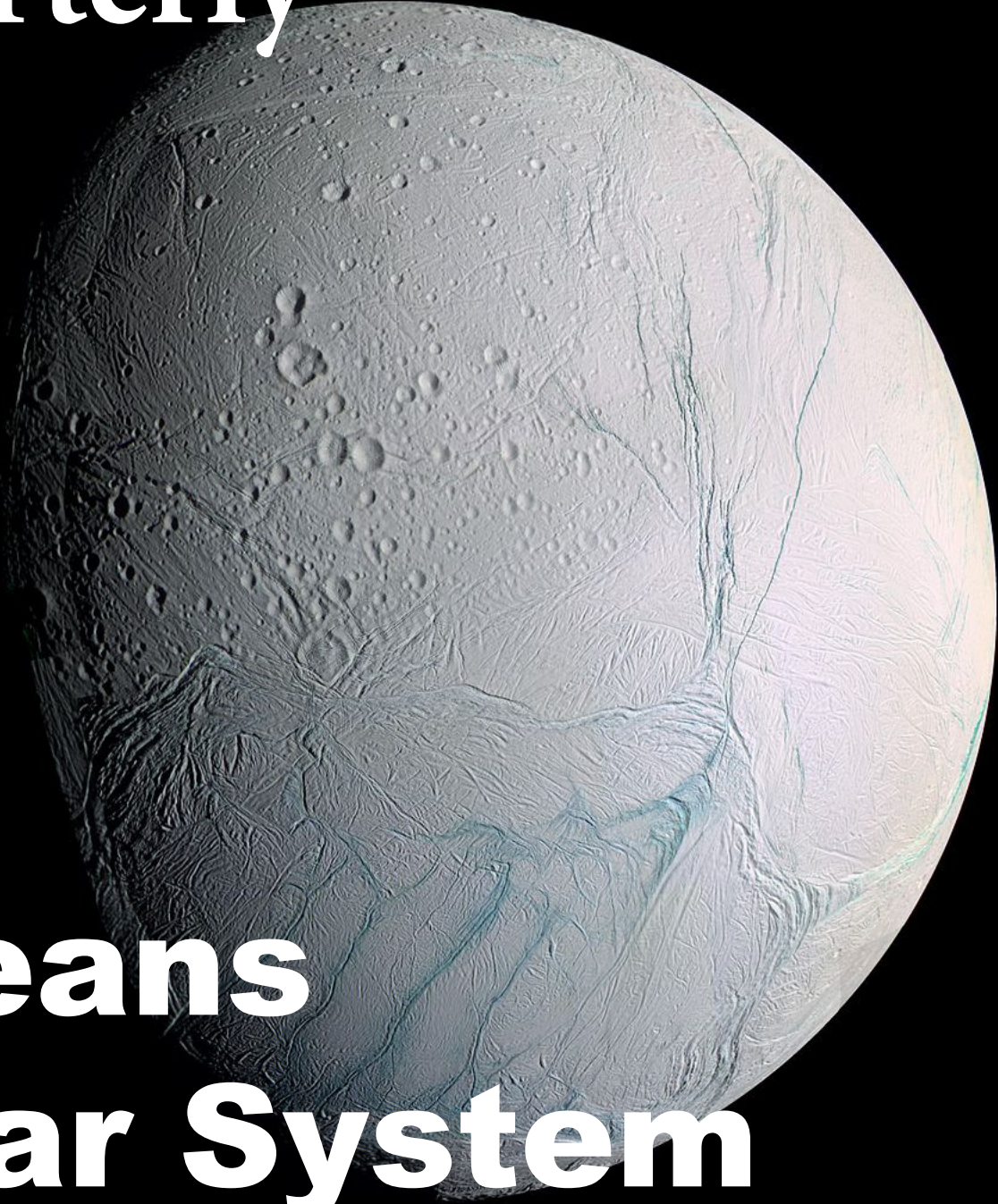
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